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Perspective

Chronic Pain Effect in Inflammatory Arthritis

Yvonne Veehof*

Department of Rheumatology, Brigham and Women's Hospital, Boston, Massachusetts, United States

DESCRIPTION

Pain is the most common reason people with arthritis see a rheumatologist. Patients consistently consider pain as one of their most priorities, and pain is the single most important factor in determining the patient's overall assessment of disease activity. Although pain is often interpreted as a sign of inflammation, the correlation between pain intensity and measures of peripheral inflammation is not perfect. The prevalence of chronic non-inflammatory pain syndromes such as fibromyalgia is higher in patients with arthritis than in the general population. Patients with fibromyalgia arthritis have higher measures of disease activity and lower quality of life than inflammatory patients without fibromyalgia. Chronic pain affects 116 million people, more than the sum of those affected by diabetes, heart disease and cancer. In the United States, the painful costs of lost productivity and treatment costs exceed $635 billion annually. Pain is the main reason people with arthritis seek out a rheumatologist. Among Rheumatoid Arthritis (RA) patients, 68%-88% rated pain as one of their top three priorities and 90% rated pain as one of their top three priorities. The American College of Rheumatology's Pain Management Task Force recently stated that "pain is perhaps the most important outcome that rheumatologists report."

Inflammatory peripheral pain

Historically, pain in patients with arthritis was caused by peripheral inflammation. Treatment with Disease-Modifying Anti-Rheumatic Drugs (DMARDs) is effective in reducing symptoms of inflammatory pain, with mean pain visual analog scores (VAS 0-100) decreasing by approximately 20-40 points in trials of new biologic agents, such as tocilizumab, and in clinical trials focusing on patients with early-stage rheumatoid arthritis.

Non-inflammatory central pain

However, despite effective treatment with DMARDs, observational studies of arthritis indicate that many patients continue to have moderate pain. In a recent study of RA patients in remission of inflammatory disease (28-joint disease activity score (DAS28) <2.6), clinically significant pain frequency (numerical score of pain ≥ 4) is 12%. These findings suggest a non-inflammatory component of pain, such as structural joint damage, pain from other etiologies, and/or dysregulation of central pain-regulating mechanisms, as occurs in generalized chronic pain states, such as fibromyalgia.

One of the most common sites for non-inflammatory pain is the back. Few studies have examined the prevalence of back pain in RA patients. In a study of 1076 RA patients and 1491 community controls, the reports that the prevalence of chronic back pain was 19% among RA patients and 25% among controls. These results suggest that, although chronic back pain is common among RA patients, it is not more prevalent among RA patients than healthy controls.

In contrast, the prevalence of common non-inflammatory conditions, such as fibromyalgia, is significantly higher in RA than in the general population. 13% to 25% of RA patients have fibromyalgia, and an additional 7% to 15% have widespread chronic pain that does not meet the criteria for fibromyalgia. In a study of 1,487 patients with early-stage arthritis from the Canadian Early Arthritis Cohort (CATCH), the incidence of fibromyalgia was highest in the year after being diagnosed with arthritis, with cumulative incidence of 6.77 per 100 persons in a year. Predictors of fibromyalgia include severe pain, poor mental health, and absence of cyclic citrullinated peptide antibodies. This study suggests that the development of chronic non-inflammatory pain occurs early in the disease process and that moderate to severe pain can induce central nervous system sensitization, which is associated with other conditions chronic pain in general.

The mechanism for the development of generalized chronic pain in patients with arthritis is poorly understood. Trial studies on pain sensitivity have shown that the threshold for pressure (pressure causes pain) is lower in RA patients than in healthy controls. These thresholds are lower at joint and non-articular sites, suggesting that changes in central pain-regulatory mechanisms, such as loss of central pain regulation and

Correspondence to: Yvonne Veehof, Department of Rheumatology, Brigham and Women's Hospital, Boston, Massachusetts, United States, E-mail: yveeh@partners.org

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sensitivity, may impair mediators of generalized pain in RA. Conditioned pain dysregulation is the impairment of opioidergic and/or serotonergic-noradrenergic allergen dimerization pathways that normally mediate analgesia, whereas central sensitization is defined as an increase excitation of the pain-transmitting neurons of the central nervous system. Conditional pain ataxia is reduced or absent in patients with chronic non-inflammatory pain, such as fibromyalgia, irritable bowel syndrome, and osteoarthritis, while central sensitization is enhanced. Studies are underway to elucidate their involvement in arthritis.

Pain is a significant problem in patients with arthritis. It affects measures of disease assessment and quality of life. Peripheral inflammation is a common cause of pain in inflammatory arthritis, but other factors, including non-inflammatory central pain mechanisms, may also contribute to pain sensation. Larger longitudinal studies, involving quantitative sensory and functional neuroimaging, are needed to elucidate the involvement of central pain mechanisms in rheumatoid arthritis. It is important to determine whether these mechanisms differ according to the type of inflammatory arthritis. Till date, mostly research has focused on rheumatoid arthritis, and very few have looked at the impact of pain in other types of inflammatory arthritis.